

In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 21-924V

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CHRISTIAN M. GATTO,

* Chief Special Master Corcoran

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Petitioner,

* Filed: February 28, 2025

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v.

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SECRETARY OF HEALTH
AND HUMAN SERVICES,

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Respondent.

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Phyllis Widman, Widman Law Firm, LLC, Linwood, NJ, for Petitioner.

Nina Ren, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION¹

On February 12, 2021, Christian Gatto filed a petition for compensation under the National Vaccine Injury Compensation Program (the “Vaccine Program”).² Petition (ECF No. 1). Petitioner alleges that two meningococcal vaccines he received on February 13, 2019, caused his Guillain-Barré syndrome (“GBS”).

In February 2024, I set a briefing schedule for a ruling on the record. *See* Scheduling Order, dated Feb. 12, 2024. The parties have filed their respective briefs. Petitioner’s Brief, dated June 29, 2024 (ECF No. 57) (“Br.”); Respondent’s Opposition, dated July 26, 2024 (ECF No. 60) (“Opp.”); Petitioner’s Reply, dated Sept. 30, 2024 (ECF No. 62) (“Reply”). Now, based on the parties’ filings and the record, I deny entitlement. Petitioner has not preponderantly demonstrated

¹ Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction “of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy.” Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its present form. *Id.*

² The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter “Vaccine Act” or “the Act”]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

that the meningococcal vaccines he received could cause GBS.

I. Factual Background

On February 13, 2019, Petitioner (then 16 years old) received two versions of the covered meningococcal vaccine during a routine well-child visit at AdvoCare Haddonfield Pediatrics (“AdvoCare”) in Haddonfield, New Jersey.³ Ex. 3 at 8–11. There is no record evidence of any immediate reaction to this vaccination event (despite the receipt of two vaccines at once).

Two weeks later, on February 28, 2019, Mr. Gatto’s mother called AdvoCare and reported that he had begun to experience chills, a bad headache, and possible fever that very morning. Ex. 3 at 24. (No mention was made of the vaccination, or any developing symptoms in the interval between this medical visit and the date of vaccination). Petitioner was seen that same day, and he reported a frontal headache, warmth, congestion, and a slight cough. *Id.* at 5. Petitioner had a fever (101.1°F) and nasal discharge, but did not test positive initially (via rapid testing) for certain viral infections, including influenza A and B. *Id.* at 5, 7. Petitioner was diagnosed with acute frontal sinusitis and prescribed a five-day course of antibiotics. *Id.* at 6.

Nine days later (March 9, 2019), Mr. Gatto’s father called AdvoCare reporting that Petitioner had been experiencing a “bad [headache] since this [morning.]” Ex. 3 at 25. Petitioner was seen the next day, and he reported additional complaints such as runny nose, cough, decreased appetite and activity, nausea, vomiting, muscle aches, and fatigue. *Id.* at 2. On exam, he was congested and had mild redness in his mouth. *Id.* Petitioner now tested positive for and was diagnosed with influenza A, and was prescribed a five-day course of Tamiflu, an antiviral medication. *Id.* at 3.

Evidence of Neurologic Symptoms

On March 12, 2019, Petitioner’s father told AdvoCare treaters that Petitioner had not stopped vomiting, was weak and dehydrated, had “constant tingling in his feet,” and had stopped taking Tamiflu. Ex. 3 at 26. That evening, Mr. Gatto was taken to the emergency room, where he reported nausea, weakness, fevers, and a generalized dull aching moderate pain that was associated with chills, loss of appetite, and vomiting. Ex. 4 at 10. He exhibited malaise and weakness, and his abdomen was mildly tender throughout and “crampy,” but his lower extremities were normal, and he had no abnormal neurologic findings. *Id.* at 12. The ER provider ordered intravenous pain medication and fluids. *Id.*

³ Petitioner received the Bexsero (meningococcal conjugate for serogroup B) and Menactra (meningococcal conjugate vaccine for serogroup A, C, and Y) vaccines, both of which are contained in the Vaccine Injury Table. See 42 C.F.R. § 100.3(a).

Petitioner subsequently underwent an abdominal and pelvic CT for suspected appendicitis, but imaging “demonstrate[d] mesenteric adenitis”⁴ and “[p]ossible bibasilar pneumonia.” Ex. 4 at 18–19, 22. His laboratory results revealed elevated white blood cell count and elevated liver function, but rapid Group A streptococcus and respiratory panel tests yielded negative results. *Id.* at 20, 33–35. Petitioner was also at this time now displaying diminished reflexes in his lower extremities, and had urinary retention. *Id.* at 23. The ER provider suspected GBS, adding that “[Petitioner] had no recent vaccinations however a recent viral-like syndrome.” *Id.* The provider transferred Mr. Gatto to St. Christopher’s Hospital for Children (St. Christopher’s Hospital) in Philadelphia, PA, that same day. *Id.*

At St. Christopher’s Hospital, the admitting provider recorded that Petitioner had displayed “symptoms of sinusitis” in February 2019, which resolved after treatment, but that he subsequently developed congestion and a runny nose. Ex. 5 at 986. Petitioner had also experienced fevers, abdominal pain, and vomiting, receiving one dose of Tamiflu for treatment. *Id.* Petitioner subsequently developed bilateral leg weakness, which worsened until he could no longer walk, and his current complaints included constipation, headaches, and continued abdominal pain and vomiting. *Id.*

Based on petitioner’s abnormal physical exam, the admitting provider diagnosed Petitioner with suspected GBS, but could not “rule out . . . [a] post infectious proc[]ess, or systemic illness causing [petitioner’s] symptoms.” *Id.* at 986–89. Similarly, the attending neurologist who saw Petitioner noted that he had experienced a “recent illness approximately [two] weeks ago when he developed [upper respiratory infection (URI)] symptoms with sinusitis and was treated with antibiotics,” that “[h]e also rec[ei]ved vaccine [sic] including HPV [sic] at his pediatrician’s visit,” and that he had presented with bilateral leg weakness “that started [a] few day[s] ago with a prece[]ding viral illness [one] week ago.” *Id.* at 1006, 1008.

While hospitalized that March, Petitioner was started on intravenous immunoglobulin for presumed atypical GBS. Ex. 5 at 1234, 1265–67. On March 15, 2019, an infectious disease specialist saw Petitioner, and offered the conclusion that Petitioner had experienced an “[a]cute onset of lower extremity weakness with respiratory failure in the setting of recent febrile illness.” *Id.* at 1000, 1005. The specialist explained that “GBS can be associated with recent respiratory or [gastrointestinal] infections,” including from *Campylobacter jejuni* (*C. jejuni*), cytomegalovirus, and Epstein–Barr virus (EBV). *Id.* at 1005; *see also id.* at 1266 (neurology assessment noting “[a]pproximately two-thirds of cases of acute inflammatory demyelinating polyradiculoneuropathy follow an infection, usually viral but sometimes mycoplasmal or bacterial ([C. jejuni]). The association of [C. jejuni] with [GBS] is interesting because these patients tend to

⁴ “Mesenteric Adenitis” is defined as “a syndrome characterized by right lower quadrant pain secondary to an inflammatory condition of mesenteric lymph nodes. This condition is classically mistaken for acute appendicitis due to their similarity in presentation.” *Mesenteric Adenitis*, National Library of Medicine, <https://www.ncbi.nlm.nih.gov/books/NBK560822/> (last visited Feb. 28, 2025).

develop acute motor axonal neuropathy.”). The specialist ordered additional testing that produced negative results. *Id.* at 1148, 1568–69, 1575–77 (noting that Petitioner was not tested for *C. jejuni*). On March 16, 2019, the attending neurologist concluded that “given that [Petitioner] has elevated cells with lymphocitic [sic] predominance, a viral etiology [wa]s most likely causing an atypical GBS.” Ex. 5 at 1257; *see also id.* at 1005, 1259–60 (noting Petitioner’s cerebrospinal fluid results were consistent with GBS).

Mr. Gatto was discharged from St. Christopher’s Hospital on April 8, 2019. Ex. 5 at 1016–20. His subsequent records document his rehabilitation and recovery, and he was able to walk “independently without any support” by April 25, 2019, returning to school on April 29, 2019. Ex. 2 at 3; Ex. 3 at 29, 31. By approximately three months post-vaccination, i.e., late May 2019, Petitioner was “doing fantastically well,” and no longer had “any pain or numbness or tingling in his arms or feet anymore.” Ex. 2 at 3, 5, 8. Subsequently, Petitioner continued being monitored by nephrology for his blood pressure, without material changes. *Id.* at 9 (August 6, 2019), 11 (October 29, 2019). No subsequent medical record evidence relevant to this case was filed.

II. Expert Reports

A. Petitioner’s Experts

1. Dr. Georges Ghacibeh — Dr. Ghacibeh, a neurologist and medical professor, prepared two written reports on Petitioner’s behalf. Report, dated Mar. 9, 2022, filed as Ex. 12 (ECF No. 29-1) (“First Ghacibeh Rep.”); Report, dated Nov. 18, 2022, filed as Ex. 22 (ECF No. 36-1) (“Second Ghacibeh Rep.”).

Dr. Ghacibeh received his medical degree from the Lebanese University, Faculty of Medical Sciences, in Beirut, Lebanon in 1997, and a Masters of Science in Clinical Investigation from the University of Florida in 2007. Curriculum Vitae, filed as Ex. 13 (ECF No. 29-2) (“Ghacibeh CV”) at 1. He then completed internships at Lebanese University and Staten Island University, followed by his residency in Neurology at New York University, Bellevue Medical Center. Ghacibeh CV at 1. Thereafter, Dr. Ghacibeh completed fellowships in Behavioral and Cognitive Neurology, and Clinical Neurophysiology at the University of Florida. *Id.* He is currently an Assistant Professor in the Department of Neurology at Seton Hall-Hackensack Meridian School of Medicine, as well as Chief of the Division of Neurology and Medical Director of the Primary Stroke Center at Pasack Valley Medical Center and Hackensack Meridian Health. *Id.* He is board certified by the American Board of Psychiatry and Neurology and the American Board of Clinical Neurophysiology. *Id.* at 2.

First Report

Dr. Ghacibeh's first report summarized Petitioner's medical history in a manner consistent with the records discussed above. *See generally* First Ghacibeh Rep. at 2–3. He deemed the chronologic history of Petitioner's post-vaccination development of GBS to fit his theory of the meningococcal vaccine's causal role. *Id.* at 3. He also briefly reviewed GBS as an illness, stressing that it is understood to be a peripheral nerve demyelinating disease mediated by a cross-reactive autoimmune process, sometimes associated with other (albeit distinguishable) vaccines such as the COVID-19 vaccine. *Id.* at 3–4.

Vaccines, Dr. Ghacibeh contended, are thought capable of instigating GBS through different biologic mechanisms. One general theory involves molecular mimicry occurring due to similarity between a vaccine's antigens and self-nerve protein structures/sequences, with antibodies generated in reaction to the foreign antigens cross-reacting against the mimicked self-structures, resulting in autoimmune damage. *Id.* at 4. Alternatively, “overstimulation and alteration of the normal regulation of the immune system” could occur in reaction to a vaccine, leading to dysregulation that could similarly result in an autoimmune attack (although Dr. Ghacibeh offered no independent evidence for this possible mechanism). *Id.*

To support the contention that the meningococcal vaccine specifically could cause GBS, Dr. Ghacibeh offered passive surveillance reporting (derived from the Vaccine Adverse Event Reporting System (“VAERS”) database) of several instances of GBS observed to have occurred in the wake of receipt of the meningococcal vaccine. First Ghacibeh Rep. at 3; *Guillain-Barré Syndrome Among Recipients of Menactra Meningococcal Conjugate Vaccine—United States, June – July 2005*, 40 MMWR⁵ 1023 (2005), filed as Ex. 15 (ECF No. 29-4) (the “2005 MMWR Report”). He cited no other medical or scientific studies that would support causation, however.

Petitioner's medical history was also consistent with vaccine causation in Dr. Ghacibeh's view, given the absence of other explanations. Petitioner did not initially test positive for any infectious explanation for his symptoms, and treatments specific to a bacterial infection, like antibiotics, were ineffective. First Ghacibeh Rep. at 4. By March 10–12, 2019, treaters proposed he had an influenza A infection, and yet a more precise PCR test⁶ did not confirm the existence of this viral infection. *Id.* Nor did a respiratory panel establish the existence of some other infection. *Id.* And the timeframe for Petitioner's onset matched what was known generally about GBS onset. *Id.* at 5; T. Myers et al., *Adverse Events Following Quadrivalent Meningococcal Diphtheria*

⁵ “MMWR” stands for “Morbidity and Mortality Weekly Report.”

⁶ “Polymerase Chain Reaction” is defined as “a laboratory technique for rapidly producing (amplifying) millions to billions of copies of a specific segment of DNA, which can then be studied in greater detail.” *Polymerase Chain Reaction (PCR)*, National Human Genome Research Institute, <https://www.genome.gov/genetics-glossary/Polymerase-Chain-Reaction-PCR> (last visited Feb. 28, 2025).

Toxoid Conjugate Vaccine (Menactra) Reported to the Vaccine Adverse Event Reporting System (VAERS), 2005–2016, 40 Vaccine 6291, 6293 (2020), filed as Ex. 14 (ECF No. 29-3) (describing VAERS reports involving an onset of symptoms of GBS ranging from 2 to 33 days after vaccination in persons 11–43 years of age).

Second Report

Dr. Ghacibeh’s second written report responded to the opinions offered by both of Respondent’s experts. In it, he summarily repeated his prior description of the medical record chronology. Second Ghacibeh Rep. at 2–3. However, he also maintained that Respondent’s experts had effectively proposed that Petitioner experienced *two* post-vaccination illnesses: frontal sinusitis as of February 28, 2019 (when Petitioner first presented to a treater after vaccination), and then an influenza A infection in early March (which he maintained Respondent’s experts deemed the cause of Petitioner’s GBS). *Id.* at 1.

In reaction, Dr. Ghacibeh noted again what testing revealed, as well as his views on the relationship between Petitioner’s non-neurologic symptoms and his later-diagnosed GBS. He maintained (although the medical record is not consistent with this contention) that Petitioner’s sinusitis symptoms were underway well before he was taken for treatment on the 28th, while emphasizing that at this time Petitioner tested negative for Influenza A. *Id.* at 2. But Dr. Ghacibeh questioned the sinusitis diagnosis, both due to lack of improvement despite receipt of antibiotics plus the absence of testing confirmation (like imaging). He thus opined that Petitioner’s initial symptoms were simply part of an overall monophasic process leading to GBS. *Id.*

Dr. Ghacibeh expressed doubt about later testing results that seemed to confirm the presence of an Influenza A infection. Petitioner twice tested negative for it—both early on in his post-vaccination treatment as well as in March. *Id.* at 2. And Dr. Ghacibeh doubted these testing results were due to a cessation of viral shedding attributable to the impact from treatment. Dr. Ghacibeh also again noted that the more accurate PCR test cast doubt on the rapid test positive result. *Id.* Given the above, Dr. Ghacibeh concluded that the overall sequence of events favored the meningococcal vaccine as causal of Petitioner’s GBS. *Id.* at 3.

Dr. Ghacibeh also attempted to defend that aspect of his opinion involving the “can cause” entitlement element. He argued, for example, that even if VAERS data was made up solely of passive surveillance reporting incidents, any medical or scientific study also involved “retrospective data collection,” making those kinds of studies no more reliable. Second Ghacibeh Rep. at 3. The risk of GBS after meningococcal vaccine was non-zero (even if negligible), thus allowing for the possibility of causation. *Id.* And even though scientific understanding about the association between GBS and other bacterial infections (such as *Campylobacter jejuni*) was greater than what Petitioner could show with respect to the meningococcal vaccine, the Respondent’s own experts had themselves failed to offer mechanistic evidence of their favored cause (Influenza

A)⁷—and it was likely that any cause of GBS under the circumstances had the same mechanistic explanation. *Id.* at 3–4.

2. Dr. Lawrence Steinman — Dr. Steinman is a well-credentialed⁸ neurologist and immunologist, and he has offered two written reports in this matter in support of Petitioner’s claim. Report, dated July 30, 2023, filed as Ex. 24 (ECF No. 48-1) (“First Steinman Rep.”); Report, dated Jan. 27, 2024, filed as Ex. 55 (ECF No. 54-1) (“Second Steinman Rep.”).

First Report

After several pages devoted to recounting his professional history and accomplishments, a summary of Petitioner’s medical history pertinent to the claim, and a brief recounting of the other expert reports filed as of that time, Dr. Steinman entered into a discussion of the core elements of his opinion. *See generally* First Steinman Rep. at 1–7. He agreed with the propriety of the GBS diagnosis, but maintained that the meningococcal vaccine was capable of causing it. *Id.* at 7–8.

Much of Dr. Steinman’s opinion is identical to what he has commonly offered in other Program cases, including his explanation for how molecular mimicry between a foreign antigen (whether in a vaccine or a pathogenic viral/bacterial infection) and self-protein sequences or tissue structures can result in an autoimmune cross-reaction against the self-sequences/structures, as antibodies produced in reaction to the foreign antigens mistakenly also attack self. *See, e.g.*, First Steinman Rep. at 9 (reproducing same *Scientific American* diagram that appears in almost all of Dr. Steinman’s reports).⁹ He also endeavored to opine as to the number of amino acids in a peptide sufficient for a mimicking cross-reaction to occur (First Steinman Rep. at 10–11)—although this case does not turn on that question—and noted as well that *distinguishable* demyelinating diseases like multiple sclerosis (“MS”) have been linked to other wild viral infections—in particular the Epstein-Barr Virus (“EBV”) (*Id.* at 11–14).

In order to prove in this case that the mechanism of molecular mimicry between components of the meningococcal vaccine and self-nerve structures capable of leading to GBS

⁷ Of course, it almost goes without saying that *it is not Respondent’s burden* (except in cases where the burden shifts) to affirmatively prove that another factor caused the injury, rather than the purported vaccine, and in so doing provide preponderant evidence of a likely mechanism—even to do so could strengthen Respondent’s objections to a claim.

⁸ While I note that a CV for Dr. Steinman was not filed in the instant matter, I am very familiar with Dr. Steinman’s experience and background as a neurologist and immunologist, given how often he testifies in the Vaccine Program.

⁹ *See, e.g., White v. Sec’y of Health & Hum. Servs.*, No. 20-1319V, 2023 WL 4204568, at *4 n.8 (Fed. Cl. Spec. Mstr. June 2, 2023), *mot. for review den’d*, 168 Fed. Cl. 660 (2023), *appeal docketed*, No. 2024-1372 (Fed. Cir. Jan. 23, 2024); *J.S. v. Sec’y of Health & Hum. Servs.*, No. 16-1083V, 2022 WL 20213038, at *8 (Fed. Cl. Spec. Mstr. July 15, 2022), *mot. for review den’d*, 164 Fed. Cl. 314, *redacted opinion issued*, No. 16-1083V, 2023 WL 1956306 (Fed. Cl. Feb. 13, 2023), *aff’d*, No. 2023-1644, 2024 WL 4051281 (Fed. Cir. Sept. 5, 2024).

was a reasonable explanation for vaccine causation in this matter, Dr. Steinman utilized his commonly-adopted approach of conducting a BLAST¹⁰ search to look for similarities between one component of the meningococcal vaccine—the diphtheria *toxin*—and “two paranodal antigens targeted in GBS,” contactin and neurofascin. First Steinman Rep. at 15. (Importantly, it should be noted that the meningococcal vaccine is intended to protect against a *bacterial* infection, and primarily includes polysaccharide components of different strains of the bacterium. Menactra Package Insert, filed as Ex. A-6 (ECF No. 33-7) (“Menactra Insert”); Bexsero Package Insert, filed as Ex. A-7 (ECF NO. 33-8). Since polysaccharides are not constructed of amino acid peptide sequences, a showing of homologic amino acid sequence similarity would not be possible. Thus, Dr. Steinman’s theory zeroed in on the diphtheria conjugate also included in the vaccine to boost its immunogenicity, ignoring the primary purpose of the vaccine. In addition, the vaccine contains a less-pathogenic *toxoid* version of diphtheria toxin, further distinguishing it from the basis for Dr. Steinman’s analysis. Menactra Insert at 2.¹¹

Reliable medical and scientific studies showed that proteins located at junctions along the myelin sheath were an attack point in GBS, Dr. Steinman maintained. First Steinman Rep. at 15–17; J. Fehmi et al., *Nodes, Paranodes and Neuropathies*, 1 J Neurol Neurosurg Psychiatry 61 (2018), filed as Ex. 47 (ECF No. 52-23). Dr. Steinman’s BLAST search was able to identify sufficient amino acid sequential homology between the diphtheria toxin and neurofascin and contactin, protein components of myelin. First Steinman Rep. at 18–22. He deemed the results to be statistically significant, given the standard applied by earlier, foundational research into autoimmune disease propagated with molecular mimicry as its mechanism, and otherwise consistent with a reliable analytic methodology. *Id* at 22–24.

Dr. Steinman also endeavored to bulwark his causation theory with other evidence. He contrasted “comforting” epidemiologic studies undermining a meningococcal vaccine-GBS association with the Centers for Disease Control’s (“CDC”) own observations of temporal associations between receipt of the vaccine and development of GBS. First Steinman Rep. at 25–26; W. Yih et al., *No Risk of Guillain-Barré Syndrome found after Meningococcal Conjugate Vaccination in Two Large Cohort Studies*, 12 Pharmacoepidemiol. Drug Saf. 1359 (2012), filed as Ex. C-21 (ECF No. 33-40) (“Yih”). But even if in most cases the vaccine was safe for administration, “the risk is not zero.” First Steinman Rep. at 26. Dr. Steinman otherwise offered

¹⁰ “Basic Local Alignment Search Tool (“BLAST”) is a medical/scientific internet resource that aids in “finding regions of similarity between biological sequences. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance.” *BLAST*, National Library of Medicine, <https://blast.ncbi.nlm.nih.gov/Blast.cgi> (last visited Feb. 28, 2025).

¹¹ Dr. Steinman nevertheless referenced the diphtheria “toxin” throughout his first written report. *See generally* First Steinman Rep.; *see also* R. Raju et al., *Epitopes for Human CD4+ Cells on Diphtheria Toxin: Structural Features of Sequence Segments Forming Epitopes Recognized by Most Subjects*, 12 Eur J Immunol 3207 (1995), filed as Ex. 49 (ECF No. 52-25).

no evidence supporting a link between the meningococcal vaccine/wild bacterial infection and GBS.

Besides opining on causation, Dr. Steinman attempted to bulwark Dr. Ghacibeh's contention that it was unlikely there was an alternative infectious explanation for Petitioner's injury. He emphasized, for example, that Petitioner's rapid test confirming the existence of an influenza A infection was not later corroborated by subsequent, more reliable testing. First Steinman Rep. at 7–8. Thus, there were not credible alternative explanations for why Petitioner developed GBS. And Petitioner's first neurologic symptom (leg weakness) was noted in the medical record to have occurred on March 12, 2019—27 days post-vaccination, thereby falling within the 3–42-day timeframe recognized as medically acceptable for post-vaccination GBS. *Id.* at 26.¹²

Second Report

Dr. Steinman's second report endeavored to rebut a number of contentions made by Respondent's primary immunologic expert, Dr. Andrew MacGinnitie. First, despite his erroneous references to the vaccine's diphtheria "toxin" component in his earlier report, Dr. Steinman maintained that he fully understood that the meningococcal vaccine contained only the *toxoided* version—but argued that this was a meaningless distinction, since the toxoid still included the same peptide sequences capable of sparking a putative autoimmune cross-reaction due to molecular mimicry (as reflected by literature he had filed in the matter). Second Steinman Rep. at 1–2. Thus, the conjugate component of the meningococcal vaccine remains "highly immunogenic" even if no longer toxic. *Id.* at 2.

Second, Dr. Steinman went to great lengths to defend his BLAST search homology determinations as reliable and meaningful. Second Steinman Rep. at 2–8. In so doing, he contended that some of the studies he relied upon (which he acknowledged involved intentionally-artificial contexts, including this use of powerful adjuvants never incorporated in actual vaccines) did establish a scientifically-reliable number of amino acids necessary for a possible autoimmune cross-reactive attack leading to disease. *Id.* at 2–3. He argued that more recent studies (particularly involving MS and EBV) confirmed that a pentamer (or five amino-acid peptide sequence) would be sufficient for a cross-reaction. *Id.* at 4. He proposed that his database search methodology used to identify these homologic similarity sequences was reliable and sound. *Id.* at 4–6. And he reiterated the contention from his first report that homology specific to two paranodal nerve

¹² To support the evidentiary sufficiency of this timeframe, however, Dr. Steinman referenced an article often cited in Vaccine Program cases where vaccine-caused GBS is alleged. L. Schonberger et al., *Guillain Barré Syndrome following Vaccination in the National Influenza Immunization Program*, 100 Am. J. Epidemiology 105 (1979), filed as Ex. 54 (ECF No. 52-30) ("Schonberger"). Of course, Schonberger involves the *flu vaccine* (and in fact a version of that vaccine not wholly consistent with what individuals receive today, since Schonberger is a 45-year-old article), and there is otherwise no agreed-upon timeframe in which *all* vaccines arguably would cause GBS.

proteins (contactin and neurofascin) was highly relevant herein, since they were likely attack foci for cross-reacting autoantibodies (although he again invoked research specific to EBV, acknowledging at the same time that the nerve proteins he offered as target antigens “do not belong to the EBV family”). Second Steinman Rep. at 7.

B. Respondent’s Experts

1. Dr. Gregory Holmes—Dr. Holmes—like Dr. Ghacibeh, a neurologist and medical professor—prepared a single written report for Respondent. Report, dated Aug. 2, 2022, filed as Ex. C (ECF No. 33-19) (“Holmes Rep.”).

Dr. Holmes attended Washington and Lee University for his undergraduate degree, and the University of Virginia School of Medicine for his medical degree. Curriculum Vitae, filed as Ex. D (ECF No. 33-49) (“Holmes CV”) at 1. He then completed an internship in Pediatrics at Yale University School of Medicine, followed by his residency in Pediatric Neurology at the University of Virginia School of Medicine. Holmes CV at 1. Dr. Holmes is currently a Professor and Chair of Pediatrics and Neurological Sciences at Larner College of Medicine, University of Vermont. *Id.* at 2. He also acts as Physician Leaders at Neurology Health Care Service, University of Vermont Medical Center. *Id.* at 1, 3. Dr. Holmes is board certified by the American Board of Pediatrics, the American Board of Psychiatry and Neurology with Special Competence in Child Neurology, as well as the American Board of Clinical Neurophysiology. *Id.* at 2.

Dr. Holmes began his opinion with an overview of Petitioner’s relevant medical history. *See generally* Holmes Rep. at 2–10. He agreed that Petitioner had been properly diagnosed with GBS. *Id.* at 11. But Dr. Holmes maintained that certain aspects of the medical chronology helped demonstrate why vaccine causation was unlikely. In particular, Dr. Holmes emphasized, onset of Petitioner’s neurologic symptoms came after he reported a variety of flu-like symptoms, including headache, rhinorrhea, cough, and nausea. *Id.* at 10–11. A rapid test performed before Petitioner’s March hospitalization suggested the existence of an Influenza A infection, and GBS was known to often be preceded by viral or bacterial infections. *Id.* at 11; Z. Karalok et al., *Guillain-Barré Syndrome in Children: Subtypes and Outcome*, 11 Childs Nerv Syst. 2291 (2018), filed as Ex. C-2 (ECF No. 33-21). Indeed, Dr. Holmes maintained, two-thirds of GBS patients reported some antecedent viral or bacterial infection, with a number of different infections having been associated with GBS. *Id.*; Y. Hao et al., *Antecedent Infections in Guillain-Barré Syndrome: A Single-Center, Prospective Study*, 12 Ann. Clin. Transl. Neurol. 2510 (2019), filed as Ex. C-4 (ECF No. 33-23).

Other aspects of Petitioner’s history were inconsistent with vaccine causation, Dr. Holmes maintained. Dr. Ghacibeh had proposed that Petitioner’s most GBS-consistent symptoms began two weeks after he first experienced more flu-like symptoms (which themselves began no sooner than two weeks post-vaccination), but Dr. Holmes deemed this scenario to be “highly unlikely.”

Holmes Rep. at 14. If Petitioner's initial symptoms had been reflective of some vaccine-related reaction, like malaise, they should have appeared within a few days of vaccination, not two weeks later. *Id.*; C. Ouandaogo et al., *Adverse Events following Immunization during Mass Vaccination Campaigns at First Introduction of a Meningococcal A Conjugate Vaccine in Burkina Faso, 2010*, *Vaccine* 46 (2012), filed as C-23 (ECF No. 33-42). And some of Petitioner's initial symptoms, like rhinitis, were inconsistent with vaccine malaise in any event. Holmes Rep. at 14.

Dr. Holmes rejected Dr. Ghacibeh's opinion that the meningococcal vaccine could be causal of Petitioner's GBS. Reliance on passive surveillance reporting of instances of post-vaccination GBS was misguided, Dr. Holmes contended, given that this kind of data only provides evidence of a temporal relationship rather than actual causation. Holmes Rep. at 13; R. Davis, *Vaccine Safety Surveillance Systems: Critical Elements and Lessons Learned in the Development of the US Vaccine safety Datalink's Rapid Cycle Analysis Capabilities*, 1 *Pharmaceutics*. 168 (2013), filed as Ex. C-18 (ECF No. 33-37). In fact, other studies that had more carefully evaluated this same kind of passive surveillance reporting evidence, but applying different methodologies, had deemed there to be no safety signal at all between the meningococcal vaccine and peripheral neuropathies like GBS. R. Li et al., *Meningococcal Conjugate Vaccine Safety Surveillance in the Vaccine Safety Datalink using a Tree-Temporal Scan Data Mining Method*, 4 *Pharmacoepidemiol. Drug Saf.* 391 (2018), filed as Ex. C-19 (ECF No. 33-38) ("Li").

Li's authors relied on passive surveillance reports of adverse events among a pool of teenagers (enrolled in large managed care health providers in several states) who received a meningococcal vaccine dose between 2005 and 2014 (more than 1.2 million doses). From this database, and applying a "tree-temporal data mining method" (Li at 392), they identified signals suggestive of meaningful risks only with respect to three categories of injuries manifesting within six weeks of vaccination: "diseases of the skin and subcutaneous tissue," nonspecific kinds of health concerns like fever or allergic reactions, or diseases of the respiratory system. *Id.* at 394, 395–96. Li deemed the first two categories "expected outcomes following vaccination," and suggested the third was a false signal, with respiratory-related issues likely attributable to comorbid factors. *Id.* at 395. GBS was not one of the reported adverse events. *Id.* at 394, Table 1.

Other scientific/medical studies had attempted more directly to gauge whether the meningococcal vaccine can cause GBS, but had not so concluded. Holmes Rep. at 13; P. Velentgas et al., *Risk of Guillain-Barré Syndrome after Meningococcal Conjugate Vaccination*, 12 *Pharmacoepidemiol. Drug Saf.* 1350 (2012), filed as Ex. C-20 (ECF No. 33-39) ("Velentgas"); see also Yih at 1359.¹³ Velentgas was a study commissioned in reaction to the findings of the 2005 MMWR Report—filed by Dr. Ghacibeh in support of causation in this case. That report had observed five VAERS reports of GBS in young adults, beginning within two to four weeks of

¹³ Yih is a letter to the editor of the relevant medical journal discussing the findings of Velentgas, and deeming them significant.

vaccination. Velentgas at 1350–51; 2005 MMWR Report. To evaluate whether the 2005 MMWR Report suggested a possibility of causation, Velentgas’s authors conducted a retrospective study, obtaining data from an initial pool of twelve million teenagers and young adults vaccinated between 2005 and 2008. *Id.* at 1350–52; *see also Id.* at 1355 (deeming the pool of subjects “one of the largest cohorts ever assembled for an epidemiologic study”). From that original pool, 1.4 million instances of receipt of the meningococcal vaccine were identified, but only 99 cases of GBS (meeting the study’s diagnostic criteria) in periods of up to six-weeks post-vaccination were seen. *Id.* at 1354. In fact, *no instances* were observed within 42 days of vaccination. *Id.* Overall, Velentgas identified no increased risk of GBS after receipt of a meningococcal vaccine. *Id.* at 1357.

Dr. Holmes also expressed doubt that scientific understanding of the relationship between GBS and a different bacterial infection (*Campylobacter jejuni*) suggested the same was true for the meningococcal vaccine’s bacterial antigenic components. Medical science had confirmed the former relationship through a number of studies—including an animal study where direct “immunization” of the antigenic mimics into animal subjects resulted in GBS-like symptoms, thus establishing scientific awareness of the specific molecular mimicry at issue. N. Yuki et al., *Carbohydrate Mimicry between Human Ganglioside GM1 and Campylobacter Jejuni Lipooligosaccharide Causes Guillain-Barré Syndrome*, 31 Proceedings Nat. Academy Sci. U.S.A. 11404 (2004), filed as Ex. C-29 (ECF No. 3348). Nothing comparable was understood about the meningococcal bacterium.

2. Dr. Andrew MacGinnitie — Dr. MacGinnitie is a pediatric immunologist, and he provided Respondent with two written expert reports. Report, dated Aug. 8, 2022, filed as Ex. A (ECF No. 33-1) (“First MacGinnitie Rep.”); Report, dated Oct. 16, 2023, filed as Ex. E (ECF No. 51-1) (“Second MacGinnitie Rep.”).

Dr. MacGinnitie received his medical degree from the University of Chicago Pritzker School of Medicine, graduating with both an M.D. and a Ph.D. from the Department of Pathology. Curriculum Vitae, filed as Ex. B (ECF No. 33-18) (“MacGinnitie CV”). Thereafter he completed a residency in Pediatrics in the Boston Combined Residency Program, training at Boston Children’s Hospital and Boston Medical Center, followed by an Allergy/Immunology fellowship at Boston Children’s Hospital. MacGinnitie CV at 2. Dr. MacGinnitie maintains an active clinical practice, and has extensive experience in caring for children and adults with a variety of immunologic diseases, including reactions to vaccines. *Id.* He is board certified in both Allergy/Immunology and Pediatrics, and is a Fellow of the American Academy of Allergy, Asthma and Immunology. *Id.* Additionally, he performs research and has published articles in several areas related to immunology, including proposed vaccine reactions and primary immunodeficiency. *Id.* At the time of the filing of the CV in this case, Dr. MacGinnitie was as Associate Professor at

Harvard Medical School, and an attending physician at Boston Children’s Hospital.¹⁴ *Id.* at 1-2.

First Report

Dr. MacGinnitie’s first report reacted only to Dr. Ghacibeh’s initial written opinion. Although Dr. MacGinnitie provided a summary of the relevant medical history (consistent with the other experts), he did not purport to offer an opinion on diagnosis. First MacGinnitie Rep. at 1-4). But Dr. MacGinnitie disputed Dr. Ghacibeh’s contention that the meningococcal vaccine could cause GBS, taking issue with several different facets of Petitioner’s causation theory.

First, Dr. MacGinnitie noted that Dr. Ghacibeh had offered no “specifics” with respect to the meningococcal vaccine that would show how the same molecular mimicry mechanism believed to link other bacteria, like *C. jejuni*, to GBS would also apply to the meningococcus bacterium. First MacGinnitie Rep. at 5. Absent some scientific evidence that meningococcal antigens had a demonstrated cross-reactive potential with any nerve-associated structures, it was wholly speculative to conclude that this particular vaccine could trigger GBS. *Id.* This was especially so given how common in nature it was for different molecular protein sequences or human tissue structures to have similarity with foreign antigens. *Id.* at 5-6. And homologic/structural molecular similarity was in any event insufficient by itself to establish causality. *Id.* at 6.¹⁵

Second, Dr. MacGinnitie denied that relevant medical and scientific evidence *could* support an association between GBS and the meningococcal vaccine. Like Dr. Holmes, he emphasized that VAERS data was not particularly probative of causation. First MacGinnitie Rep. at 9. At the same time, other reliable evidence (such as Velentgas) strongly undermined the existence of any causal relationship. *Id.*

In addition (and consistent with Dr. Holmes), Dr. MacGinnitie noted how Petitioner’s actual presentation did not appear to support vaccine causation (while supporting an infectious trigger). *See generally* First MacGinnitie Rep. at 6-8. Thus, Dr. MacGinnitie reiterated Dr. Holmes’s observation that many of Petitioner’s pre-neurologic presenting symptoms were not consistent with vaccine-associated malaise (and had occurred too long thereafter as well). *Id.* at 6. But Dr. MacGinnitie also endeavored to bulwark the conclusion that Petitioner’s GBS was caused by an influenza A infection, despite testing results that did not (initially at least) confirm the

¹⁴ I am aware from my work in other cases that Dr. MacGinnitie has since left to work at a hospital in Milwaukee, Wisconsin, but an updated CV for him was not filed in this matter.

¹⁵ Dr. MacGinnitie deemed Dr. Ghacibeh’s briefly-referenced alternative mechanism (in which a vaccine’s components could result in immunologic dysregulation, leading to autoimmune harm) no better substantiated. First MacGinnitie Rep. at 6. But because Dr. Steinman’s causal explanation appears to set forth what Petitioner primarily relies upon in asserting a causation theory, I do not deem this somewhat-offhand proposal from Dr. Ghacibeh to require in-depth analysis.

presence of that infection.

To that end, Dr. MacGinnitie argued that (a) Petitioner's first symptoms at the end of February 2019 appeared reflective of a viral infection, (b) he tested positive for Influenza A virus, and (c) Influenza A was known to be associated with GBS. First MacGinnitie Rep. at 6–7; L. Grimaldi-Bensouda et al., *Guillain-Barré Syndrome, Influenza-like Illnesses, and Influenza Vaccination during Seasons with and without Circulating A/H1N1 Viruses*, 174 Am J Epidemiol 326 (2011), filed as Ex. A-8 (ECF No. 33-9); H. Lehmann et al., *Guillain-Barré Syndrome after Exposure to Influenza Virus*, 10 Lancet Infect Dis (2010), filed as Ex. A-9 (ECF No. 33-10). Dr. MacGinnitie disputed Dr. Ghacibeh's contention that the rapid test likely elicited a false positive result, as well as the argument that subsequent PCR testing (which was negative) was reliable, since by this time the Petitioner was likely not shedding viral particles in sufficient amounts to be detectable (especially given the medications he had been receiving). *Id.* at 7; F. Hayden et al., *Use of the Oral Neuraminidase Inhibitor Oseltamivir in Experimental Human Influenza*, 2828 JAMA 1240 (1999), filed as Ex. A-10 (ECF No. 33-11).

And even if it were true that Petitioner had not experienced such an Influenza A infection, the medical record still allowed for the conclusion that he had experienced *some form* of respiratory infection, given his symptoms and treater views. In fact, the receipt of antibiotics by themselves constituted a risk factor for GBS. L. Levison et al., *Association of Hospital-Diagnosed Infections and Antibiotic Use with Risk of Developing Guillain-Barré Syndrome*, 96 Neurology e831 (2021), filed as Ex. A-13 (ECF No. 33-14). And again, Petitioner's symptoms in total (neurologic plus all the earlier concerns) were not consistent with GBS, while Petitioner's mesenteric adenitis¹⁶ (identified on March 13, 2019) was known to be specifically associated with viral infections. R. Helbling et al., *Acute Nonspecific Mesenteric Lymphadenitis: More than “No Need for Surgery,”* 2017 BioMed Research International 1 (2017), filed as Ex. A-14 (ECF No. 33-15).

Second Report

Dr. MacGinnitie's second written report responded primarily to Dr. Steinman's causation theory, which was more fleshed out than what Dr. Ghacibeh had provided. Dr. MacGinnitie noted that Dr. Steinman's opinion centered around an attempt to show amino acid sequence homology between the meningococcal vaccine's diphtheria toxoid component (an inert form of the *toxin*—which Dr. Steinman's report seemed to erroneously assume was actually in the vaccine) and certain nerve cell proteins. Second MacGinnitie Rep. at 1–2. But he deemed the theory deficient in several regards.

¹⁶ “Mesenteric Adenitis” is defined as “right lower quadrant pain secondary to an inflammatory condition of mesenteric lymph nodes. This condition is classically mistaken for acute appendicitis due to their similarity in presentation.” *Mesenteric Adenitis*, National Library of Medicine, <https://www.ncbi.nlm.nih.gov/books/NBK560822/> (last visited Feb. 28, 2025).

First, Dr. MacGinnitie contended, Dr. Steinman had invoked medical and scientific studies that were not useful in predicting that any vaccine could prompt an autoimmune response via molecular mimicry. Second MacGinnitie Rep. at 2–4. Studies in which five identical amino acids could be identified as sufficient for a molecular mimicry-based mechanism to “work” all involved artificial experimental conditions (such as the employ of highly powerful experimental adjuvants) not comparable to the conditions in which a person receives a vaccine. *Id.* at 2, 3. Some articles involved direct injection of pertussis *toxin*, which the meningococcal vaccine does not include. *Id.* at 3. And others involved distinguishable conditions (multiple sclerosis), or diseases mediated by T-cells, whereas the theory proposed herein was that B-cell-instigated antibodies were causal (consistent with the very purpose of the vaccine: to create antibodies). *Id.* at 3.

Dr. Steinman also had not shown sufficient homology between the compared peptide sequences in the diphtheria toxoid and proposed nerve protein component peptides. Second MacGinnitie Rep. at 3. In fact, in nature “consecutive stretches of 5–9 identical amino acids are common between microbial and human proteins,” and yet do not result in disease. Second MacGinnitie Rep. at 5; *Institute of Medicine, Adverse Effects of Vaccines: Evidence and Causality* (K. Stratton et al., eds. 2012). Dr. Steinman further relied on outdated literature to defend the significance of his findings. Second MacGinnitie Rep. at 4. And Dr. MacGinnitie contended that BLAST searches he had performed, in reaction to Dr. Steinman’s earlier searches, identified “no human sequences with notable homology to diphtheria toxoid.” See *BLAST Search: Homo Sapiens and Contactin-1 and Neurofascin*, filed as Ex. E-9 (ECF No. 51-10). The fact that the diphtheria toxoid was capable of eliciting *some* immune response was not meaningful, since “the purpose of vaccination is to generate an immune response.” Second MacGinnitie Rep. at 4.

Second, Dr. MacGinnitie noted that Dr. Steinman’s other evidence was unsupportive of causation. Research showing that EBV and MS might be associated (via the mechanism of molecular mimicry) was inapposite, since (a) the timeframe for post-infectious development of MS was significantly longer than GBS (years rather than weeks), (b) a live viral infection was inherently more immunogenic than a vaccine, causing collateral harm in the body that would further disease processes in a manner a vaccine could not, and (c) studies exploring this association noted that other pathologic processes (in particular, “hypermutation”) not implicated by vaccination were thought to play a role in why molecular mimicry was a relevant mechanism for that context. *Id.* 5–6. Indeed, all of the above only underscored in Dr. MacGinnitie’s view “how stringent the protections that exist for the human immune system against autoimmunity” are, since so many immunologic safeguards would need to fail before cross-reactivity was likely to occur. *Id.* at 6.

By contrast, Dr. MacGinnitie highlighted the epidemiologic evidence (referenced in his first report, and also discussed in part by Dr. Holmes) that was unsupportive of a vaccine-GBS relationship. Second MacGinnitie Rep. at 6. In so doing, he stressed that some studies not only

showed no meaningful risk from GBS after administration of the meningococcal vaccine, but also that one study (Velentgas) in fact found *no* instances of post-meningococcal vaccine GBS, in a sample of over two million doses—contradicting Dr. Steinman’s argument that “zero risk” had not been established. And since Dr. Steinman’s theory was reliant on a meningococcal vaccine component (diphtheria toxoid) common to other vaccines, it would be reasonably expected that an increased rate of GBS after receipt of other kinds of toxoid-containing vaccines should be evident—yet studies did not so find. J. Tuttle et al., *The Risk of Guillain-Barré Syndrome after Tetanus-Toxoid-Containing Vaccines in Adults and Children in the United States*, 87 Am J Public Health 2045 (1997), filed as Ex. E-14 (ECF No. 51-15) (“Tuttle”).

3. Dr. Hayley Gans — Dr. Gans is a pediatric infectious disease specialist, and she prepared a single written report for Respondent. Report, dated Oct. 21, 2023, filed as Ex. F (ECF No. 51-16) (“Gans Rep.”).

Dr. Gans is a Clinical Professor in the Department of Pediatrics and Division of Pediatric Infectious Disease at Stanford University. Curriculum Vitae, filed as Ex. G (ECF No. 51-30) (“Gans CV”) at 1. She received her medical degree from SUNY at Syracuse and completed a residency and fellowship at Stanford University. Gans CV at 1. In her clinical capacity, Dr. Gans has cared for hundreds of infants and children with infections and has been involved with disease prevention through mitigation and immunizations. Gans Rep. at 1. She currently conducts immunology research in the field of infectious diseases and studies vaccine responses in several populations including normal hosts, HIV-infected children, premature children, children who have received organ transplants and children with autoimmune diseases. *Id.* Dr. Gans serves on several regulatory boards overseeing the safety of vaccines and is involved with case adjudication for vaccine studies. She is board certified in both Pediatrics and Pediatric Infectious Diseases. Gans CV at 2.

As with the other experts in this case, Dr. Gans’s report includes her own summary of Petitioner’s medical history. *See generally* Gans Rep. at 2–7. But she emphasized several points throughout. She deemed Petitioner’s presentation on February 28, 2019, to be more consistent with a respiratory illness than a post-vaccination, malaise-like reaction. *Id.* at 2. Indeed, these symptoms in her view began too long after vaccination to be associated with that earlier event. *Id.* at 12. Petitioner’s March treatment visits were similarly less akin to a vaccine reaction than an unspecified “influenza-like illness.” *Id.* at 3. Further, in the March period before Petitioner first manifested neurologic symptoms, he experienced things that could not be deemed to be GBS-associated, such as abdominal complaints. *Id.* And treaters thereafter consistently attributed his GBS to a febrile illness—and this was so even though no specific infectious agent had been identified. *Id.* at 4–5.¹⁷

¹⁷ Dr. Gans also argued that Dr. Ghacibeh misinterpreted the records in places—maintaining, for example, that Petitioner’s symptoms from February 28, 2019, to early March reflected a monophasic course (in which he did not

Taking into account the relevant history, Dr. Gans deemed it most likely that Petitioner's GBS was attributable *either* to the respiratory illness he first complained of in late February 2019, or some unspecified gastrointestinal illness that began in March. Gans Rep. at 6. GBS is understood to have an association with antecedent infections—although only half the time can the infection be specifically identified (in part due to the fact that the GBS manifests *after* the infectious process ends). *Id.* at 7; B. van den Berg et al., *Guillain-Barré Syndrome: Pathogenesis, Diagnosis, Treatment and Prognosis*, 8 Nat Rev Neurol. 469 (2014), filed as Ex. F-5 (ECF No.51-21). Nevertheless, a number of different viral and bacterial agents have been identified as capable of causing GBS—including Influenza A. Y. Hao et al., *Antecedent Infections in Guillain-Barré Syndrome: A Single-Center, Prospective Study*, 12 Ann Clin Transl Neurol 2510 (2019). But in Dr. Gans's understanding, only those infectious processes that result in production of “specific antibody responses” capable of cross-reacting with existing nerve component peptide sequences or structures will cause it. Gans Rep. at 8. Here, Petitioner's history of respiratory and GI symptoms prior to GBS onset was consistent with such an infectious cause (while the direct symptoms at issue were not characteristic of GBS). *Id.* at 11.

Vaccines, by contrast, are less commonly associated with GBS, Dr. Gans maintained. Gans Rep. at 8. Medical science has only reliably associated the swine flu version of the flu vaccine from the 1970s with GBS, but otherwise “no evidence of a true association exists” for any other modern versions. *Id.* She identified a study from the past ten years that she maintained rejected a GBS causal association with several commonly-administered vaccines. R. Baxter et al., *Lack of Association of Guillain-Barré Syndrome with Vaccinations*, 2 Clin. Infect. Dis. 197 (2013), filed as Ex. F-7 (ECF No. 51-23) (“Baxter”).

The same was true, Dr. Gans argued, for the meningococcal vaccine, as reflected by several studies. Gans Rep. at 9. One post-licensure study found no GBS signals (simply based on reported instances of post-vaccination GBS) for vaccine formulations containing a meningococcal component. J. Hansen et al., *Post-Licensure Safety Surveillance Study of Routine Use of Quadrivalent Meningococcal Diphtheria Toxoid Conjugate Vaccine*, 35 Vaccine 6879 (2017), filed as Ex. F-8 (ECF No. 51-24) (“Hansen”) (concluding “[t]his study did not detect any safety concerns following [Menactra] and provides reassurance that [Menactra] administered as part of routine care was not associated with unexpected safety risks.”). Dr. Gans (like Respondent's other two experts) also referenced Velentgas, underscoring the extent to which that study found that the incidence of post-meningococcal vaccination GBS did not exceed the likely background rate. Gans Rep. at 9; Velentgas at 1354.¹⁸

improve despite treatment), when in fact the record suggested Petitioner's sinusitis had resolved (but was replaced thereafter by a second illness with slightly different symptomatic features). Gans Rep. at 10–11, 12–13.

¹⁸ Dr. Gans also noted that forms of the meningococcal vaccine administered abroad had also not been found associated with GBS. Gans Rep. at 9; G. Hall et al., *Post-Licensure Observational Safety Study after Meningococcal B Vaccine*

Dr. Gans rejected the scientific reliability of VAERS data, as invoked by Drs. Ghacibeh and Steinman, in establishing causality. Gans Rep. at 9. These kinds of reports did not rely on confirmed instances of GBS diagnoses, and otherwise only established temporal relationships with vaccination. *Id.* at 9–10. At best, passive reports provided signals that could be the basis for more methodologically rigorous study—and in fact this had occurred with respect to the form of meningococcal vaccine at issue herein. *See generally* 2005 MMWR Report. The 2005 MMWR Report actually found that the rate of reported instances of post-vaccination GBS could be attributed to chance alone, when considered in conjunction with the background rate. Gans Rep. at 10. A larger study from 2020 that utilized VAERS data reached roughly the same conclusion. T. Myers et al., *Adverse Events following Quadrivalent Meningococcal Diphtheria Toxoid Conjugate Vaccine (Menactra®) Reported to the Vaccine Adverse Event Reporting System (VAERS)*, 38 Vaccine 1, 7 (2020), filed as Ex. C-14 (ECF No. 33-33) (“identif[ying] 144 reports of GBS, of which 24 were excluded due to alternative diagnoses or no evidence of GBS.”)

III. Procedural History

This case has been pending for approximately four years. After Respondent’s notice in January 2022 that he intended to defend against the claim (ECF No. 25), the parties began the process of filing expert reports. Expert discovery was completed with the filing of Dr. Steinman’s second report in January 2024. Thereafter, I determined that the matter could be resolved via ruling on the record, and the parties filed their respective briefs. The matter has been ripe for resolution since late September 2024.

IV. Parties’ Arguments

Petitioner

Petitioner argued that he has presented a reliable and persuasive medical theory of causation linking the meningococcal vaccines to his GBS. Br. at 8.¹⁹ First, Petitioner’s experts “do not support the notion that Petitioner was simply ill with the influenza virus, which then caused [his] GBS.” *Id.* at 9. Not only did Dr. Ghacibeh note that Petitioner’s antibiotic course failed to improve his condition, but he had established that some of the testing Petitioner received was unreliable. *Id.*; *see also* B. Nivin & T. Zancocchio, *Cluster of False-Positive Influenza B Virus Rapid Antigen Test Results in a New York City Hospital*, 50 J Clin Microbiol 3114 (2012), filed as Ex. 19 (ECF No. 29-8) (highlighting the sensitivity of influenza rapid tests and noting sensitivity rates as 60% or less when compared to viral culture or PCR).

¹⁹ *4CMenB (Bexsero) Vaccination within the Routine UK Immunisation Program*, 24 Vaccine 3296 (2021), filed as Ex. F-9 (ECF No. 51-25).

¹⁹ I note that Petitioner’s experts do not contend that receipt of two meningococcal-containing vaccines at the same time multiplied his risk, or caused harms beyond what one could allegedly cause.

In addition, even if a flu virus could trigger GBS, Dr. Ghacibeh established that “the same mechanism that can cause GBS from an influenza virus is precisely what caused Petitioner’s GBS from the meningococcal vaccines.” Br. at 11. Dr. Steinman took that a step further, offering a theory “showing that the components of the Menactra vaccine contain peptide sequences aligning with paranodal proteins like contactin and neurofascin that can trigger GBS.” *Id.* at 12; Steinman First Rep. at 27. Thus, based on the BLAST searches, case reports, and the filed medical literature, Petitioner had met his burden of establishing that the meningococcal vaccine can cause GBS. Br. at 13.

Petitioner also contended that he had identified a logical sequence of cause and effect between the meningococcal vaccines and his development of GBS sufficient to meet the second, “did cause” prong. Br. at 13. He emphasized Dr. Ghacibeh’s observation that “the entire respiratory panel performed at Kennedy was negative also [which] suggests that the symptoms that [Petitioner] was experiencing were unlikely to be due to a viral illness and more likely to be related to complications of the meningococcal [v]accinations.” *Id.*; Ghacibeh First Rep. at 3. Dr. Steinman had similarly opined that “[t]here is no better clear alternate cause” for Petitioner’s development of GBS. Br. at 14. Moreover, and according to Petitioner, there is not enough record evidence to support a diagnosis of a flu infection, and thus, the proposition that his immune system overreacted when exposed to the vaccines, resulting in GBS, has preponderantly been established. *Id.*

Lastly, Petitioner contended that he established a medically acceptable temporal association between the meningococcal vaccinations and his GBS. Br. at 14. Relying on previous reports supporting an onset of GBS between two- and thirty-three-days post-vaccination, Dr. Ghacibeh opined that the timeline of Petitioner’s receipt of the meningococcal vaccines, his subsequent development of URI symptoms, and then his development of GBS was consistent with what medical science suggested would be reasonable. *Id.*; Ghacibeh First Rep. at 5. Dr. Steinman in particular deemed an onset within 12 days of vaccination to be medically acceptable. Br. at 14; Steinman First Rep. at 27.

Respondent

Respondent argued that Petitioner cannot establish causation. First, Respondent maintained, Petitioner had failed to produce reliable evidence that the meningococcal vaccine can cause GBS—noting that much of the available epidemiology fails to support such a relationship. Opp. at 12 (emphasis added). In support, Respondent cited to the Velentgas and Yih articles, which demonstrate little to no association with an increased GBS risk. *Id.*; Velentgas at 1 (concluding the Menactra vaccination was not associated with an increased GBS risk); Yih at 1 (noting that “if there is an increased risk of GBS associated with [Menactra], it is likely to amount to less than 0.66 excess case per million adolescents vaccinated.”).

Similarly, Dr. Steinman’s reliance on studies involving the diphtheria toxin component of the Menactra vaccine were undercut by epidemiologic findings. Opp. at 13–14; *see also* Baxter at 197, 201 (study of more than thirty million individuals between 1995 and 2006 finding no increased GBS risk with tetanus-diphtheria vaccines); Tuttle at 4 (concluding that “the number of cases of [GBS] observed after administration of such vaccines in both adults and children [wa]s less than the number expected by chance alone.”). Moreover, Petitioner’s use of case reports and findings from passive surveillance databases, such as VAERS, and merely invoking the theory of molecular mimicry but without corroborating it with other evidence, did not amount to a preponderant showing. Opp. at 15–18.

Next, Respondent maintained that Petitioner failed to demonstrate that the meningococcal vaccines did cause his GBS. Opp. at 19. Medical science recognized the “frequency of clinical symptoms associated with respiratory and abdominal infections preceding a diagnosis of GBS and the linkage of these infections with specific antibodies that cross-react with gangliosides,” leaving “little doubt of the direct role infections have in causing GBS.” *Id.* at 20 (citing Gans Rep. at 8). And Petitioner’s contemporaneous medical records established “[two] well-documented clinical infections” which manifested twelve and three days prior to Petitioner’s GBS onset. Opp. at 20. While Respondent acknowledged that statements of treating physicians are not binding, he nonetheless noted that several of Petitioner’s treaters provided “reasoned support for a viral etiology for [P]etitioner’s GBS.” *Id.* at 21; *see also* Ex. 4 at 22; Ex. 5 at 986–89, 1000–08, 1257.

Finally, Respondent contended that Petitioner had not preponderantly shown that his GBS began within a medically acceptable timeframe following his receipt of the meningococcal vaccine on February 13, 2019. Opp. at 22. To support onset, Petitioner had relied on VAERS and various case reports, as well as articles specific only to the flu vaccine, but without further explanation as to why the studied influenza strain was relevant to the vaccines at issue herein. *Id.* at 23.

V. Applicable Law

A. Standards for Vaccine Claims

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a “Table Injury”—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a “Non-Table Injury”). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also* *Moberly v. Sec’y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec’y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).²⁰

²⁰ Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec’y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec’y of Health & Hum. Servs.*, 59 Fed. Cl. 121,

In this case, Petitioner cannot assert a Table claim (as there is no such claim with respect to the meningococcal vaccine and GBS).

For both Table and Non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was “not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): “(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury.”

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a “reputable medical theory,” demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner’s theory must be based on a “sound and reliable medical or scientific explanation.” *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be “legally probable, not medically or scientifically certain.” *Knudsen*, 35 F.3d at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed “not through the lens of

124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

the laboratorian, but instead from the vantage point of the Vaccine Act’s preponderant evidence standard.” *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245.

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has *consistently rejected* the contention that it can be satisfied merely by establishing the proposed causal theory’s scientific or medical *plausibility*. See *Kalajdzic v. Sec’y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments “for a less than preponderance standard” deemed “plainly inconsistent with our precedent” (citing *Moberly*, 592 F.3d at 1322)); *Boatman v. Sec’y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *LaLonde v. Sec’y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (“[h]owever, in the past we have made clear that simply identifying a ‘plausible’ theory of causation is insufficient for a petitioner to meet her burden of proof” (citing *Moberly*, 592 F.3d at 1322)); *see also Howard v. Sec’y of Health & Hum. Servs.*, 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) (“[t]he standard has been preponderance for nearly four decades”), *aff’d*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec’y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* “addresses the petitioner’s overall burden of proving causation-in-fact under the Vaccine Act” by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner’s medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec’y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine “did cause” injury, the opinions and views of the injured party’s treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 (“medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a ‘logical sequence of cause and effect show[s] that the vaccination was the reason for the injury’”) (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec’y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec’y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (“there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and

cannot be rebutted”). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians’ conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically-acceptable temporal relationship.” *Id.* A petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder’s etiology, it is medically acceptable to infer causation.” *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one’s requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review denied*, (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. Analysis of Expert Testimony

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 594–96 (1993). See *Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). “The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.” *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually

employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) (“uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted”). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See e.g., Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner’s case. Where both sides offer expert testimony, a special master’s decision may be “based on the credibility of the experts and the relative persuasiveness of their competing theories.” *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert’s conclusion “connected to existing data only by the *ipse dixit* of the expert,” especially if “there is simply too great an analytical gap between the data and the opinion proffered.” *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for rev. denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. Appx. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert’s credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 (“[a]ssessments as to the reliability of expert testimony often turn on credibility determinations”); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) (“this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act”).

Expert opinions based on unsupported facts may be given relatively little weight. *See Dobrydnev v. Sec'y of Health & Hum. Servs.*, 556 F. Appx. 976, 992–93 (Fed. Cir. 2014) (“[a] doctor’s conclusion is only as good as the facts upon which it is based”) (citing *Brooke Group Ltd. v. Brown & Williamson Tobacco Corp.*, 509 U.S. 209, 242 (1993) (“[w]hen an expert assumes facts that are not supported by a preponderance of the evidence, a finder of fact may properly reject the expert’s opinion”)). Expert opinions that fail to address or are at odds with contemporaneous medical records may therefore be less persuasive than those which correspond to such records. *See Gerami v. Sec'y of Health & Hum. Servs.*, No. 12-442V, 2013 WL 5998109, at *4 (Fed. Cl. Spec. Mstr. Oct. 11, 2013), *aff'd*, 127 Fed. Cl. 299 (2014).

C. Consideration of Medical Literature

Both parties filed numerous items of medical and scientific literature in this case, but not every filed item factors into the outcome of this Decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (“[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision”) (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. Appx. 875, 884 (Fed. Cir. 2013) (“[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered”).

D. Standards for Ruling on the Record

I am resolving Petitioner's claim on the filed record. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

I. Program Consideration of Claims Involving Meningococcal Vaccine and GBS

There are very few reasoned Program decisions involving *any* injury and the meningococcal vaccine, let alone GBS.²¹ In one prior case, however, a special master did conclude a petitioner had succeeded in establishing that the meningococcal vaccine could cause GBS. *Whitener v. Sec'y of Health & Hum. Servs.*, No. 06-477V, 2009 WL 3007380 (Fed. Cl. Spec. Mstr.

²¹ There are in comparison many *settled* cases in which an individual received the meningococcal vaccine (alone or with other vaccines) and then developed GBS. But settlements provide no reasoned guidance for how to decide this case. At most, they suggest to claimants that a claim involving the same vaccine and injury could be resolved successfully (perhaps based on the Government's views about how to best marshal its resources in the Vaccine Program)—but not that the causation theory in question has been *conceded* by Respondent to be valid, let alone that it is scientifically reliable.

Sept. 2, 2009). The *Whitener* petitioner received the meningococcal vaccine on December 30, 2004, with onset of neurologic symptoms approximately one month later. *Whitener*, 2009 WL 3007380, at *2–3. The special master deemed causation demonstrated under the first *Althen* prong. *Id.* at *20. As grounds, *Whitener* noted that the petitioner’s expert had referenced the 2005 MMWR Report—the same item that sparked the Velentgas study several years later. *Id.* at *8, n.17. The 2005 MMWR Report was deemed to provide at least an “incidental association” sufficient to support causation, even if it included no proposed mechanistic explanation for the association. *Id.* at *20. The special master also seemed to give weight to the fact that GBS is immune-mediated (making it possible *any* vaccine could spark it, merely through immune stimulation). *Id.*

Several years later, however, *Whitener*’s reasoning was called into question. *Tompkins v. Sec’y of Health & Hum. Servs.*, No. 10-271V, 2013 WL 3498652, at *26 (Fed. Cl. Spec. Mstr. June 21, 2013) (finding petitioner did not prove several vaccines received at same time caused his GBS), *mot. for review den’d*, 117 Fed. Cl. 713 (2014). The *Tompkins* special master concluded, among other things, that the petitioner had not demonstrated the meningococcal vaccine could cause GBS, and criticized the probative value of the 2005 MMWR Report relied upon in *Whitener* (and offered in this case as well). *Tompkins*, 2013 WL 3498652, at *26. Not only had the 2005 MMWR Report expressly disclaimed that the observed incidents of post-vaccination GBS had causal significance, but since its release no subsequent scientific studies had corroborated the significance of the temporal association it observed. *Id.* (noting an absence of evidence “in the ensuing seven years of any case reports, studies, or other evidence suggesting that the spike in cases was more than coincidence”). The *Tompkins* special master also gave weight to other epidemiologic evidence suggesting no increased risk of GBS after receipt of the meningococcal vaccine (although it does not appear that Velentgas, published a year prior to *Tompkins*’s issuance, was considered). *Id.* Accordingly, although there is no obvious Program consensus for how this kind of claim should be resolved, *Tompkins*’s reasoning is far more sound and persuasive than what was evidenced in *Whitener*.

In addition, I have had repeated occasion to consider claims that the pneumococcal vaccine—analogous to the meningococcal, since neither are viral-based²²—can cause GBS, but have rejected the causation theory offered each time. See, e.g., *Gamboa-Avila v. Sec’y of Health & Hum. Servs.*, No. 18-925V, 2023 WL 6536207, at *24–31 (Fed. Cl. Spec. Mstr. Sept. 11, 2023), *mot. for review den’d*, 170 Fed. Cl. 441 (2024), *appeal docketed*, No. 2024-1765 (Fed. Cir. May 1, 2024). The causation arguments made in this matter echo what I have confronted in these past cases, but found scientifically unreliable. At bottom, theories that this sort of bacterial infection-oriented vaccine can cause peripheral nerve demyelination over-rely on what is known about the

²² The pneumococcal vaccine protects against a bacterial infection, like the meningococcal vaccine. *Bielak v. Sec’y of Health & Hum. Servs.*, No. 18-761V, 2023 WL 35509, at *11–12, 18 (Fed. Cl. Spec. Mstr. Jan. 3, 2023). In addition, because a bacterium is at issue, both vaccines attempt to stimulate an immune response through use of a conjugate additive that boosts the ability of the immune system to recognize the bacterial polysaccharide components.

association between the flu vaccine (a *viral*-aimed vaccine) and GBS, but with far less scientifically-reliable proof. And because arguments about protein components (which are made up of amino acids) cannot be applied to the bacterial segments in this kind of vaccine, experts in such cases either shift their attention to the conjugate (included in the vaccine solely to prompt an immune reaction)—even though its inclusion in the vaccine is somewhat incidental to its primary purpose—or draw strained connections between lipid myelin structures and the vaccine’s polysaccharide antigenic components, but without reliable evidence suggesting that the demyelinating process key to GBS would begin at those points. *Gamboa-Avila*, 2023 WL 6536207, at *31.

In fact, arguments that *any* vaccines are as equally causal of GBS as the flu vaccine almost always borrow wholesale from scientific evidence that is actually only preponderant *in the context of the flu vaccine*. Indeed, there has been a tendency in the Vaccine Program toward effectively broadening the existing flu vaccine-GBS Table claim to encompass virtually every other covered vaccine—even though none are also the subject of a Table claim for this kind of peripheral neuropathy. I have not been willing to do the same, and have instead evaluated the evidence specific to the vaccine at issue, to determine if in fact the preponderant standard has been met as applied to causation-in-fact claims. *Gamboa-Avila*, 2023 WL 6536207, at *25. I approach the causation theory advanced in this case in a similar manner.

II. Petitioner Has Not Carried His Burden of Proof

The parties do not dispute that Petitioner was properly diagnosed with GBS, and hence their disagreement turns on Petitioner’s success in meeting the *Althen* prongs. I find that the Petitioner’s inability to meet the first, “can cause” prong is fatal to the claim (although the second prong also was not preponderantly established).²³

Prong One

The causation theory embraced in this case almost wholly relies on the kind of showing of homology (common to expert opinions from Dr. Steinman) that I have repeatedly deemed insufficient to establish causation *by itself*. *Schultz v. Sec'y of Health & Hum. Servs.*, No. 16-539V, 2020 WL 1039161, at *22 n.24 (Fed. Cl. Spec. Mstr. Jan. 24, 2020) (“[m]ere demonstration of theoretical homology alone, based on computer-driven searches involving databases of amino acid sequences, does not carry the day”).²⁴ Proof of some amino acid sequential identity between a

²³ I only include a discussion of the first and second prongs, since a claimant must establish all three in order to prove entitlement to compensation. *Dobrydnev v. Sec'y of Health & Hum. Servs.*, 566 Fed. Appx. 976, 980 (Fed. Cir. 2014).

²⁴ My analysis herein does not include a highly-detailed review of Dr. Steinman’s reports and the literature they cite. This is because the form of report he offers replicates the reports he has filed in numerous other cases, in which he seeks to show (almost wholly on the basis of molecular mimicry) that a vaccine other than the flu vaccine could cause

vaccine's components and those of some nerve myelin component does not prove the vaccine likely is causal of injury—and yet that constitutes most of Petitioner's causation theory. Moreover, Dr. MacGinnitie noted a number of limitations to Dr. Steinman's reasoning on the likelihood of a cross-reaction due to mimicry that were not rebutted—from the fact that homology is common in nature without disease, to the degree certain experiments proving pathology as a result of mimicry are distinguishable.

Otherwise, any supporting evidence for causation that was offered was thin. Petitioner has provided nothing to show a relationship between the wild analogs to the vaccine's components and GBS—diphtheria in particular—since Dr. Steinman's entire homology-based theory could only show amino acid sequence similarity between this viral vaccine component and myelin components. Petitioner also failed to establish reason to believe that meningococcal bacterial antigens could themselves likely lead to GBS (in comparison to *other* kinds of bacteria, like *C. jejuni*, that are clearly well-associated). Evidence derived from VAERS databases to fill this evidentiary gap is not particularly probative, since (a) the reported instances of GBS contained in a VAERS report are unconfirmed, and (b) a VAERS report only establishes a temporal association between vaccination and injury. *Howard v. Sec'y of Health & Hum. Servs.*, No. 16-1592V, 2022 WL 4869354, at *21, n.15 (Fed. Cl. Spec. Mstr. Aug. 31, 2022), *mot. for review den'd, decision aff'd*, (Fed. Cl. May 18, 2023), *aff'd*, (Fed. Cir. June 7, 2024) (noting “it has been observed in the Program that VAERS data is not particularly probative of causation unless supplemented with other reliable evidence—since a VAERS reports only establishes a temporal, post-vaccination occurrence, and thus shines no light on the possibility of causation itself”). More significantly (and as noted in *Tompkins* more than ten years ago), the incidental support for causation provided in evidence like the 2005 MMWR Report, and deemed worthy of weight in *Whitener*, had been eroded by time, as no subsequent studies provided ballast for its tentative suppositions.

By contrast, Petitioner's experts ineffectively rebutted the substantial and methodologically-robust epidemiologic evidence that the meningococcal vaccine is *not* reliably associated with GBS. It is a foundational principle in Vaccine Program cases that special masters may take into account epidemiologic findings even though petitioners are not required to offer such evidence in attempting to prove causation. *Taylor v. Sec'y of Health & Hum. Servs.*, 108 Fed. Cl. 807, 819–21 (Fed. Cl. 2013) (finding special master did not err in considering epidemiological evidence). Velentgas in particular deserves evidentiary weight, since it came into being in response to the very 2005 MMWR Report that the *Whitener* special master deemed probative in favor of causation—and that Dr. Ghacibeh filed in this very action. In fact, even some studies that relied primarily on the kind of passive surveillance reporting data generated by VAERS *also* undermine the likelihood of a meningococcal vaccine-GBS association. *See generally* Li; Hansen.

GBS. *See, e.g., Gamboa-Avila*, 2023 WL 6536207, at *26–27. I am more than familiar with the kinds of opinions Dr. Steinman offers, having encountered him as an expert repeatedly over the past 11-plus years—and it is easy to tell when a “new” expert report he drafts is merely “old wine in new bottles.”

Overall, Petitioner's experts were unsuccessful in advancing a scientifically-reliable causation theory, over-relying on case reports, superseded studies, or contentions about homology untethered to other corroborative proof. Respondent's experts, by contrast, effectively identified the many weak links in the proposed theory.

Prong Two

The record does not support the conclusion that the meningococcal vaccine "did cause" Petitioner's GBS (assuming he could have met the first prong). There is no evidence of any immediate/inflammatory reaction in the two weeks after vaccination. I do not find that Petitioner's February 28, 2019, presenting symptoms reflected a vaccine reaction, as opposed to some other infectious reaction, given their sinusitis-like features. Nor does the record support the conclusion that from the end of February until March 12, 2019 (when Petitioner obtained emergency treatment) Petitioner was likely experiencing a monophasic illness that could be directly related to his vaccination.

Thus, the sole relationship between the meningococcal vaccine and Petitioner's diagnosis of GBS in March 2019 was *temporal*, with little if anything in the record suggesting an aberrant immune response was building toward manifestation of Petitioner's neurologic symptoms. This is an insufficient basis to demonstrate a "logical sequence of cause and effect" from vaccination to injury. Also significant is the fact that treaters did not propose vaccination to have been causal—while they often implicated Petitioner's pre-hospitalization infectious symptoms as more likely linked. *See, e.g.*, Ex. 5 at 986–89, 1006, 1008, 1257.

The record is in fact replete with evidence supporting treater speculation that Petitioner's GBS was due to some form of concurrent viral infection that began well prior to onset of GBS-like symptoms. Petitioner presented with infectious symptoms on two occasions prior to when he first manifested neurologic symptoms. His February 28, 2019 symptoms could not be explained as merely a vaccine "malaise"-like reaction, since they were not limited to fever or body aches, but also included upper respiratory symptoms that are not associated with vaccination malaise. Ex. 3 at 5–7, 24. Later on in March, he complained of muscle aches and fatigue *plus* gastrointestinal issues that would also not be the product of a vaccination reaction. *Id.* at 2–3, 25.

Petitioner's experts devoted some time to attempting to discredit at least one of the putative prior infections thought by Respondent's experts to be possibly causal, Influenza A, noting that testing was inconsistent as to its existence, with later and arguably more reliable forms of testing producing negative results (although Petitioner did test positive for it on March 9th). Ex. 3 at 3. These are reasonable points—but they do not rebut the clear record evidence that *some* form of infectious process was underway (and arguably two separate infections) before onset of neurologic symptoms. I can conclude from this record that Petitioner had experienced one or more infections

before his GBS onset, even if I cannot precisely identify the character of the precise trigger—and that such an infection was *more* likely causal of Petitioner’s GBS than vaccination, whatever its precise character was.

Consideration of this evidence regarding possible alternative explanations for Petitioner’s GBS is not unfairly prejudicial. It is true that Program claimants need not *disprove* an alternative cause for an alleged injury, and I do not require them to do so here. But Respondent is also not obligated to prove an alternative cause either (except when the burden shifts to him to do so). Thus, in the context of attempting to satisfy the second *Althen* prong, petitioners must at least grapple with contradictory or damaging evidence undermining their causation showing. *M.R. v. Sec’y of Health & Hum. Servs.*, No. 16-1024V, 2023 WL 4936726 (Fed. Cl. Spec. Mstr. June 30, 2023) (stating “Petitioners must make some effort to confront a record that suggests a non-vaccine explanation for an injury if they wish to prevail on the “did cause” prong, even if they are not tasked with wholly *disproving* it as part of their *prima facie* case”) (emphasis in original). They cannot walk away from such proof. It is thus wholly legitimate for a special master to evaluate that evidence, when it exists, and include it when weighing a claimant’s success on the “did cause” prong. *Winkler v. Sec’y of Health & Hum. Servs.*, No. 18-2023V, 2021 WL 6276203 (Fed. Cl. Spec. Mstr. Dec 10, 2021), *mot. for review den’d, decision aff’d*, No. 18-203V, 2022 WL 1528779 (Fed. Cl. May 13, 2022), *aff’d*, 88 F.4th 958 (Fed. Cir. 2023); *K.A. v. Sec’y of Health & Hum. Servs.*, No. 16-989V, 2022 20213037, at *31 (Fed. Cl. Spec. Mstr. Apr. 18, 2022), *mot. for review den’d, decision aff’d*, 164 Fed. Cl. 98 (2022), *aff’d*, No. 2023-1315, 2024 WL 2012526 (Fed. Cir. May 7, 2024). This evidence of Petitioner’s pre-neurologic symptoms infections undermines greatly the conclusion that the vaccine was causal.

CONCLUSION

Claimants must carry their burden of proof. Here, Petitioner had the burden of preponderantly establishing that the meningococcal vaccine can cause GBS, or did so to him. This has not been accomplished in this case. Accordingly, I deny entitlement.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.²⁵

IT IS SO ORDERED.

s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

²⁵ Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.